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Mechanical ventilation in neurocritical care setting: A clinical approach

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Neuropatients often require invasive mechanical ventilation (MV). Ideal ventilator settings and respiratory targets in neuro patients are unclear. Current knowledge suggests maintaining protective tidal volumes of 6–8 ml/kg of predicted body weight in neuropatients. This approach may reduce the rate of pulmonary complications, although it cannot be easily applied in a neuro setting due to the need for special care to minimize the risk of secondary brain damage. Additionally, the weaning process from MV is particularly challenging in these patients who cannot control the brain respiratory patterns and protect airways from aspiration. Indeed, extubation failure in neuropatients is very high, while tracheostomy is needed in one-third of the patients. The aim of this manuscript is to review and describe the current management of invasive MV, weaning, and tracheostomy for the main four subpopulations of neuro patients: traumatic brain injury, acute

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ischemic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage.

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Introduction

Neuro patients frequently require invasive mechanical ventilation (MV) for protecting the airways from aspiration and preventing secondary brain damage by modulating oxygen and carbon dioxide levels [1]. Unfortunately, the ideal ventilator setting and respiratory targets in neuro patients are unclear and depend also on the type of brain injury, being part of the complex brain–lung interaction [2] (Fig. 1). Current knowledge concerning MV for neuro patients suggests maintaining protective tidal volumes (V_T) (6–8 ml/kg of predicted body weight (PBW)) [3,4], rather than higher V_T (9 ml/kg of PBW or higher) as customary applied many years ago [5]. Maintaining lower V_T reduces the risk of developing pulmonary complications [3]. However, this cannot be easily applied in a neuro intensive care (ICU) setting because of the need for special care to minimize the risk of secondary brain damage [6], but applying a protective MV associated with early extubation seems to improve ventilator-free days [7]. This suggests that the protective MV strategy could also be safely applied in ICU, albeit the monitoring of carbon dioxide and oxygen remains mandatory for these patients [5]. The weaning process from MV aims to reduce ventilator parameters yielding extubation for at least 48 h [8]. Extubation failure in neuro patients reaches up to 38% [9], while tracheostomy is needed in 32%–45% of them [10,11]. Besides, the timing for performing tracheostomy is still under debate in both populations [12,13].

The aim of this manuscript is to describe and review the current MV management, weaning, and tracheostomy practice for the main subpopulations of neuro patients (Fig. 2). For this purpose, we performed a systematic search of literature, by using the following terms “MV or tracheostomy or ventilation or weaning or intubation or extubation or physiotherapy or pulmonary complications” and “AIS or TBI or brain injury or SAH or ICH or brain damage.” We also performed a search about novel studies on MV and brain injury currently recruiting (Table 1).

Traumatic brain injury

Traumatic brain injury (TBI) is a large socioeconomical and healthcare burden, with a global incidence of approximately 369 cases every 100,000 habitants [14]. The severity of brain injury widely influences the occurrence of respiratory complications as well as the need for MV and tracheostomy [15]. Hospital-acquired pneumonia is a frequent complication of TBI, particularly ventilator associated pneumonia (VAP) that can occur in 20.4% of TBI patients within 5 days (3–7 days) from intubation [16]. Patients with TBI and VAP have a longer duration of MV and ICU length of stay, but not increased mortality [16]. Moreover, TBI patients with pneumonia may show worse Glasgow Outcome Scale-Extended (GOS-E) than those without and higher hospitalization costs [17]. The higher incidence of VAP has been supposed to be related with the prehospital airway management, although this was discredited in a retrospective cohort [18].

The pathophysiology of TBI is characterized by an elevation of intracranial pressure (ICP), the reduction of cerebral perfusion pressure (CPP), followed by secondary brain damage [19,20]. Besides, changes in intrathoracic pressures, oxygenation, and arterial partial pressure of carbon dioxide ($PaCO_2$) may alter this self-regulatory mechanism, which, in TBI patients, is often already compromised [21]. TBI may be complicated with an increase of ICP over 20–22 mmHg, defined as intracranial hypertension (IH) [20,22].

Endotracheal intubation and gas exchange

In TBI, the Glasgow coma scale (GCS)$\leq$8 is generally considered the optimal threshold for endotracheal intubation to secure airways from aspiration and reduce respiratory drive [23]. Prehospital interventions include the tendency to hyperventilate TBI patients, whether indicated or not by the

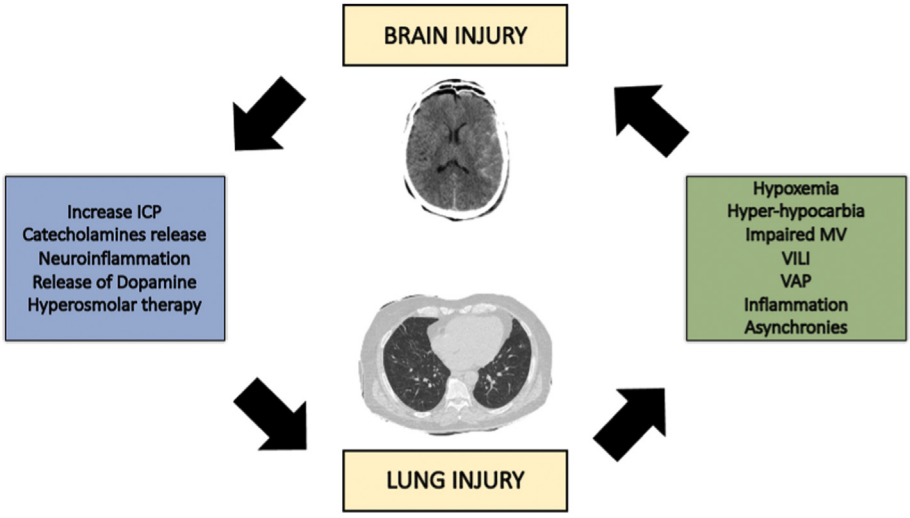

Fig. 1. Brain-lung crosstalk. Brain-lung crosstalk in neuro patients. Brain injury can influence the occurrence of lung injury by activating several mechanisms (e.g., increased ICP, the release of catecholamines, neuroinflammation, release of dopamine, and use of hyperosmolar therapies). Likewise, lung injury and MV can directly affect the brain (e.g., hypoxemia, hyper/hypocarbia, impaired MV, VILI, pneumonia, inflammation, and MV asynchronies).

guidelines [24], thus leading more frequently to hypoxia, hypotension, and acidosis [25]. This approach may deeply modify the cerebral physiology by changing the $PaCO_2$, which is considered as a major determinant of cerebral blood flow (CBF) (for CBF between 20 and 80 mmHg) [26]. Low CBF due to low $PaCO_2$ is associated with cerebral ischemia, while high CBF results in cerebral hyperemia and higher ICP [26]. TBI patients exert less CBF modifications than healthier patients because hyperventilation may lead to hypocapnia and alkalosis, redistributing oxygen and hemoglobin [27,28]. Prophylactic hyperventilation with $PaCO_2$ <25 mmHg should be avoided because of possible cerebral ischemia and

BRAIN INJURY	MV SETTING
TBI	• Intubation for GCS ≤8 • $PaCO_2$ between mmHg. Hyperventilation should be considered for 15-30 minutes with $PaCO_2$ target 30-35/32-35 mmHg in case of IH • PaO_2 >60 mmHg or SpO_2 > 94% • V_T 6-8 mL/kg of PBW • PEEP based on oxygenation and ICP • VISAGE score for tracheostomy
AIS	• Intubation for GCS ≤8 • PaO_2 >60 mmHg or SpO_2 > 94% • V_T 6-8 mL/kg of PBW • PEEP titrated on oxygenation • SET score for tracheostomy
SAH	• Intubation for incontrollable hypertension in unsecured and ruptured aneurysm, GCS≤8, GCS<2 points, optimization of MV and oxygenation, seizures control, airway protection in absence of reflexes • Follow the previous suggestions for V_T, PEEP, PaO_2 • VISAGE score for tracheostomy
ICH	• Intubation for GCS ≤8 • VT 6-8 mL/kg of PBW • PEEP titrated for oxygenation • TRACH score for tracheostomy

Fig. 2. Targets of mechanical ventilation for TBI, AIS, SAH, and ICH patients. The figure resumes all the targets to be applied in each subpopulation (TBI, AIS, SAH, and ICH) of neurocritical care patients.

cerebral infarction [5], while for short periods (15–30 min) it can be considered. Hypocapnia should be always considered in the case of refractory IH, aiming to reach $PaCO_2$ levels of 30–35/32–35 mmHg [22,29,30]. Thus, in the absence of cerebral herniation, $PaCO_2$ should be maintained between 35 and 38 mmHg [5], albeit current practice in Europe confirms that in the case of IH, the most used $PaCO_2$ target is 36–40 mmHg [31]. Respiratory rate and V_T adjustments are identified as useful tools to regulate $PaCO_2$ [5].

Oxygenation should be strictly monitored, maintaining peripheral oxygen saturation (SpO_2) >94% [30,32]. Early hyperoxia was not associated with higher inhospital mortality in a retrospective cohort of 24,148 TBI patients, while a greater risk of death was found for PaO_2<40 mmHg [33]. On the contrary, in a recent meta-analysis, hyperoxia was associated with higher mortality in MV TBI patients [34]. Final disconfirmation came from a recent small randomized controlled trial (RCT) that compared high fraction of inspired oxygen (FiO_2) (0.7) to normal FiO_2 (0.4) in MV TBI patients, which revealed that normobaric hyperoxia could be used to alleviate secondary brain ischemia without determining negative side effects. Higher FiO_2 did not increase the markers of oxidative stress, neurological injury, inflammation, and the incidence of pulmonary complications [35]. Concerning clinical practice, the most used PaO_2 is within a range of 81–100 mmHg both in the case of PaO_2/FiO_2 ratio >150 and < 150 [31]. The effects of hypocapnia and hypoxia should be monitored by using the jugular saturation of oxygen (SjO_2) or brain tissue oxygen pressure ($PbtO_2$) [5]. The individual trends of $PbtO_2$ are associated with an outcome in TBI patients [36]. In summary, in TBI patients, both hypocapnia and hypercapnia are associated with poor outcome and should be avoided.

Tidal volume and pulmonary pressures

High V_T are more often provided in brain-damaged patients than in the general critically ill population because of the need of a wider modulation of brain gases [37]. Nevertheless, MV setting in TBI patients should include protective V_T and plateau pressures [3]. As reported in a recent

Table 1
The available studies on MV practice in brain-injured patients currently recruiting or nearly recruitment on clinicaltrials.gov.

Study title	Status	Design	Primary aim	ID
Study of Variables Related to the Discontinuation of MV in Patients with Head injury (INDEXTBI)	Unknown	Prospective, observational	To evaluate the effectiveness of integrated pressure index of airway occlusion x index breathing fast and shallow (p 0.1 × f/V_T) in predicting success and failure of weaning from MV in patients with TBI	NCT01713010
The Brain and Lung Interaction (BALI) study	NR	Observational, interventional	The change in ICP as a function of PEEP	NCT04288076
Ventilator setting in patients with acute brain injury	NR	Observational, interventional	ICP increases with lung protective setting	NCT03278769
Multicenter observational study on practice of MV in brain-injured patients	NR	Prospective, observational	Description of different ventilator strategies in neuro patients admitted to ICU	NCT04459884
Inhaled nitric oxide in brain injury	R	RCT (nitric oxide, placebo)	The change in PaO_2 of 20% or greater	NCT03260569
Brain-injured patients extubation readiness study (Biper)	R	RCT (standard of care, extubation readiness clinical score)	Extubation failure in neuro patients defined as the need of reintubation or death	NCT04080440
Treatment of intracranial hypertension of severe TBI patients. Physio pathological effects of neuromuscular blocking agents (THIC Cu)	R	RCT (cisatracurium, placebo)	Area under the curve of the temporal evolution of ICP, over a period of 30 min after the administration of neuromuscular blocking agents or placebo	NCT02404779

NR, not-recruiting; R, recruiting; MV, mechanical ventilation; TBI, traumatic brain injury; V_T, tidal volume; ICP, intracranial pressure; ICU, intensive care unit; PEEP, positive end-expiratory pressure; and RCT, randomized controlled trial.

survey (VENTILO) on 687 respondents of whom 472 are from Europe, the most common V_T applied in TBI patients is 6–8 ml/kg_{PBW} in the case of PaO_2/FiO_2 >150; and 4–6 ml/kg_{PBW} in half of the cases of PaO_2/FiO_2 < 150 [31]. Among ventilator strategy, pressure-regulated volume control ventilation resulted in less fluctuation of ICP and $PaCO_2$ than the pressure controlled in TBI patients [38]. Some believe that elevated intrathoracic pressures may lead to significant changes in central venous pressure and ICP from venous congestion. A comparison between traditional ventilator mode and airway pressure release ventilation has been assessed in TBI patients, which concludes that this strategy does not significantly affect ICP and cerebral hemodynamics, being considered safe if applied [39]. Also, neutrally adjusted ventilatory assist and pressure support ventilation preserved CBF velocity in a patient recovering from brain injury, without affecting pH, $PaCO_2$, and oxygenation [40].

Positive end-expiratory pressure (PEEP) is considered as another key component of protective MV. Although PEEP may be dangerous on CBF and perfusion [41], in the case of concomitant brain and respiratory diseases, PEEP is considered safe because it results in acceptable systemic and cerebral hemodynamic changes, even when low or high (5–15 cmH_2O). A prospective study on 20 TBI patients demonstrated that increasing PEEP of up to 15 cmH_2O can improve brain tissue oxygenation [42], thus challenging the concept of low/zero PEEP (ZEEP) customarily applied for decades in neuro patients [43]. In the VENTILO survey, the mean highest PEEP is 15 cmH_2O in patients with PaO_2/FiO_2 <300 and without IH; while it is 10 cmH_2O in patients with IH [31]. The most frequent rescue strategy for refractory respiratory failure is neuromuscular blocking agents, followed by recruitment maneuvers (RMs), and prone position [31]. RMs can improve oxygenation by promoting gas exchange, although their effects on ICP could be deleterious due to the impaired jugular venous outflow and venous return. Therefore, we suggest RMs in only TBI patients in the case of severe hypoxemia under strict neuromonitoring [44].

Weaning and extubation

A positive spontaneous breathing trial test usually precedes weaning from MV. However, as neuro patients may present with impaired breathing control and respiratory driving, approximately 5%–20% of them cannot proceed to weaning and extubation [45]. Predictive factors for extubation failure in TBI patients include female sex, GCS motor score <5, moderate to large secretion volume, absent or weak cough, and MV for >10 days [46]. At present, the VISAGE score represents the most appropriate test for identifying neuro patients who can reveal extubation readiness. This score has been validated for general ICU patients, but it can also be applied in TBI patients, because specific scores have not yet been developed [47].

Before extubation is performed, chest physiotherapy is recommended for MV critically ill patients based on a multisystemic approach, because it can reduce the incidence of respiratory complications, promote weaning from MV, facilitate physical function among ICU survivors, and reduce the length of stay [48]. A study that investigated manual versus mechanical chest percussion techniques in TBI patients found that the manual technique was associated with a transient increase in ICP and impaired hemodynamics. However, the increase in ICP was transient and not clinically relevant in moderate-to-severe TBI without IH [49]. Nevertheless, chest physiotherapy did not demonstrate efficacy in the prevention of respiratory complications such as VAP, reduced MV-days, and ICU length of stay in brain-damaged patients [50].

Tracheostomy practice

Patients who have failed weaning and extubation often receive tracheostomy, although up to 79% of neuro patients do not receive an extubation attempt before tracheostomy [10]. Factors associated with the need for tracheostomy include CRASH, IMPACT, SAPS II, and APACHE II scores, age, revised trauma score, GCS, subdural hematoma, abnormal pupil reactivity, and the collapse of basal cisterns [51].

Likewise, GCS, Marshall score, chest tube, and Injury severity score have been identified as predictors for tracheostomy in TBI patients at admission. Inpatient risk factors included the requirement of an external ventricular drainage, number of operations, inpatient dialysis, aspiration, GCS on day 5, and a need for reintubation [52].

Tracheostomy timing in TBI patients is still under debate. Several definitions for early and late tracheostomy have been proposed, thus leading to misinterpretation for daily clinical management. Although general critically ill patients frequently receive tracheostomy 14–16 days after ICU admission, early tracheostomy (<15 days) demonstrated benefits in terms of ICU length of stay and MV-free days [53]. Early (within 3 days from admission) versus late (after 3 days) tracheostomy has been investigated in 98 TBI patients. Early tracheostomy reduced the ICU length of stay, antibiotic use, cost of hospitalization and pneumonia rates. No improvement in mortality was found [54]. In a meta-analysis based on eight RCTs concerning early tracheostomy (within 10 days from injury) versus late (>10 days) for TBI patients, ICU and hospital length of stays, duration of MV, and pneumonia rates decreased in the early group [55]. Very early and early tracheostomy (cutoff of 72 h from ICU admission) were also investigated. Thirty days mortality was 3% and 8%, respectively, for the very early and early group, without differences in the adverse event rate, incidence of pneumonia, and unnecessary tracheostomy rate between groups. When tracheostomy is performed within 72 h of admission, it may decrease the duration of MV and ICU length of stay [56]. Recently, Robba et al. investigated whether early (within 7 days from admission) versus late (more than 7 days) tracheostomy timing is preferred for a better patient management in terms of outcome, in a cohort of 433 tracheostomized TBI patients. Age, GCS $\leq$8, thoracic trauma, hypoxemia, and unreactive pupil have been identified as predictive factors for tracheostomy. Moreover, early tracheostomy timing revealed better outcome and less length of stay than late [57]. Fast and safe decannulation from tracheostomy seems to improve the outcome, thus a recent study investigated criteria for decannulation after tracheostomy in brain-injured patients, including among those with higher sensitivity and specificity: tracheostomy tube capping, endoscopy assessment of airway patency, swallowing instrumental assessment, and the blue dye test [58].

Acute ischemic stroke

Acute ischemic stroke (AIS) is a major cause of mortality and morbidity in the adult population, which can lead to severe disability [59]. MV is often required in these patients, and the brain area involved in AIS is one of the major determinants of the etiology of impaired MV. In fact, the level of consciousness, breathing, and swallow are regulated at the central level [60,97]. As the areas commonly targeted in ischemic injury are responsible for respiratory regulation, AIS patients are particularly prone to develop pulmonary complications such as respiratory failure, acute respiratory distress syndrome (ARDS), pulmonary edema, pulmonary embolism, stroke-associated pneumonia (SAP), and pleural effusion [37]. SAP has been identified as a major determinant of respiratory failure in AIS patients, it is usually caused by a decreased level of consciousness that reduces patients' airway patency, causes swallowing dysfunction, and dysphagia [61]. SAP accounts an incidence of 3.9%–56.6% with higher occurrence particularly in ICU [62]. Several scores have been developed to predict SAP even if uncommonly applied in clinical practice, rather than biomarkers such as C-reactive protein, dysphagia, stroke severity, and CDCP criteria [63].

Endotracheal intubation and gas exchange

General recommendation for intubation is commonly guided by neurological status (i.e., GCS $\leq$8), signs of intracranial hypertension, seizures, large infarct size that involves more than 2/3 of middle cerebral artery territory, bulbar dysfunction with the inability to protect the airway, and confirmed midline shift [64]. Oxygenation should be maintained within safe ranges, and supplemental oxygen may be administered if the SpO_2 is less than 94%, while hyperbaric hyperoxia is not recommended [64,97]. Continuous monitoring of oxygenation is strongly recommended. Moreover, during the

intubation phase, 100% FiO_2 may be administered without serious adverse effects and 3 min of pre-oxygenation or a short period of high flow nasal cannula [65]. In AIS patients treated with intra-arterial mechanical thrombectomy in the anterior cerebral circulation, admission PaO_2 >120 mmHg is associated with worse functional outcome at 90 days [66].

Tidal volume and pulmonary pressures

MV setting should take into account the possibility of secondary brain damage due to hypo/hypercapnia and/or hypo/hyperoxia (i.e., hypocapnia may determine cerebral vasoconstriction increasing the risk for secondary brain ischemia) [67]. Therefore, MV should include a protective ventilator strategy (V_T of 6 ml/kg of PBW, plateau pressure less than 30 cm H_2O, and enough PEEP to avoid both overdistension and derecruitment) to reduce the risk for pulmonary complications [68], and a ventilator management focused on the strict control of oxygen and $PaCO_2$ levels to prevent the secondary brain damage [6]. However, there is no consensus regarding the best ventilatory management (including the protective ventilator strategy) for AIS patients, as no studies are available in this specific subpopulation of neuro patients [69]. In summary, PEEP levels and RMs are considered safe in AIS patients [70].

Weaning and extubation

Although MV is lifesaving in certain neurological conditions to maintain airway patency and prevent swallowing dysfunction, its long-term use is frequently associated with increased morbidity and mortality [47]. The correct timing for extubating AIS patients is still under investigation, but based on studies on general neuro population, weaning from MV should be initiated as soon as possible [8]. Generic scores for the prediction of extubation failure in neuro patients have been developed in the latest years. The most commonly applied in the general neuro ICU population is the VISAGE score that takes into consideration gag reflex, cough, deglutition, and neurological status assessed by the visual subscale of the Coma Recovery Scale Revised [47]. AIS associated dysphagia is a major determinant of extubation readiness. A study conducted on AIS patients near to weaning and extubation revealed that dysphagia was the main determinant of extubation failure in half of them, and the reintubation rate reached 21.4%. After extubation, dysphagia commonly occurs in up to 69% of general ICU patients and 93% of AIS patients [71]. The goal standard to detect swallowing dysfunction is video fluoroscopy, although fiberoptic endoscopy has been recently suggested as a valid alternative [72].

Tracheostomy practice

Tracheostomy is required in up to 45% of neuro patients [10]. Dysphagia and GCS <10; neuroimaging like hydrocephalus, brainstem lesion, intracranial hemorrhage (ICH); surgical procedure and organs function were identified as possible parameters for identifying the patients who can benefit from tracheostomy by the stroke-related early tracheostomy (SET) score [73]. Current knowledge is still uncertain about the timing for tracheostomy in AIS patients. However, a recent study identified a similar length of stay in patients with hemorrhagic stroke, AIS, or subarachnoid hemorrhage (SAH) early (within three days) or late (between 7 and 14 days) tracheostomized; while mortality was lower in the early tracheostomized group [74]. Thus, early tracheostomy should be considered in patients who fail a first extubation attempt as no evidence is available on possible negative clinical effects.

Subarachnoid hemorrhage

Specific precautions for the management of SAH is needed with respect to patients affected by other stroke subtypes (AIS and ICH), and particularly, referring to the higher risk of SAH patients for developing vasospasm or delayed cerebral ischemia [75]. Pulmonary complications occur in up to 20%

of SAH patients, with an oxygenation impairment in up to 80% of them [76,77] thus leading to worse outcome and higher mortality [75]. The independent risk factors for the development of VAP in ICH patients include >65 years, smoke, coronary heart disease, diabetes, chronic obstructive pulmonary disease, ICU hospital stay, and MV days [78].

Endotracheal intubation and gas exchange

Indication for endotracheal intubation for SAH patients includes uncontrollable hypertension in unsecured and ruptured aneurysm, unconsciousness, GCS≤8, GCS<2 points, the optimization of ventilation and oxygenation, seizures control, airway protection in the absence of reflexes [79]. Thus, as cerebral vasospasm is associated with higher cerebral oxygen extraction, CPP and blood flow need to be strictly monitored and MV should be adequate to oxygen requirements [80].

Tidal volume and pulmonary pressures

SAH patients receive MV in higher percental (47%) as compared to AIS (5%) and ICH (26%) patients [81]. Moreover, in-hospital mortality for MV stroke patients varies among subpopulations, reaching up to 47%, 61%, and 56% in IS, ICH, and SAH, respectively [82]. Status epilepticus, pneumonia, sepsis, and hydrocephalus has been associated with a higher risk for MV in SAH patients [82]. As for other kinds of brain damage, protective MV is suggested for SAH patients, keeping protective V_T (≤8 ml/kg of PBW) and plateau pressure ≤30 cm H_2O [83].

Most recent evidence confirm that PEEP could be safely used for brain-injured patients. PEEP can act on intrathoracic pressure and impair venous return and CBF without modifying ICP [84]. The concept of low/zero PEEP was challenged by the application of 0, 5, 8, and 12 cm H_2O in MV patients with SAH or other severe brain injury with different respiratory system compliances. Patients with normal compliance showed an increase in central venous pressure and jugular pressure, with reduced mean arterial pressure and CPP. After PEEP titration, ICP and CPP slightly returned to normal values thus suggesting that respiratory system compliance may be used to detect in advance the potential harmful effects of PEEP on cerebral homeostasis [85]. However, an increase in PEEP up to 20 cm H_2O may cause a decrease in CBF and mean arterial pressure [43].

Only one study investigated the role of alveolar RMs in SAH patients with ARDS. Two RMs were tested, one by applying a continuous positive airway pressure (CPAP) of 35 cm H_2O for 40 s and the second one by using pressure-controlled ventilation with a PEEP level of 15 cm H_2O and pressure control of 35 cm H_2O above PEEP for 2 min. ICP was higher and CPP was lower for the CPAP group. Moreover, arterial oxygen tension improved in the pressure-controlled group [86].

Weaning and extubation

Extubation failure often occurs in SAH patients, particularly in those with poor neurological grading. SAH patients show an extubation failure rate of 29%, in accordance with the other stroke subpopulations. The VISAGE score can be applied for SAH patients, to detect the extubation failure in advance [47]. A decreased level of consciousness can reduce patients' airway patency and cause dysphagia. Pneumonia is common in SAH patients with higher World Federation of Neurological Surgeons score, aneurysmal SAH, secondary complications, older, longer ICU stay, and intubation. Indeed, dysphagia risk was significantly associated with age, ICU length of stay, intubation, tracheostomy, vasospasm, and new stroke [87].

Tracheostomy practice

SAH patients who cannot be extubated, frequently need tracheostomy performance. Tracheostomy performance has been detected in 17% of SAH patients [81]. The median time for tracheostomy in SAH patients was 11 days after intubation, which represents the strongest predictor for tracheostomy. Late tracheostomy has been associated with pulmonary complications, venous thromboembolism, and

pneumonia [88]. Indeed, early tracheostomy was positively associated with a shorter length of stay [89]. As there are few studies regarding tracheostomy in SAH patients, we suggest following the same rules of MV management and tracheostomy in the general population.

Intracerebral hemorrhage

ICH is considered as a medical emergency that requires urgent therapeutic management, because up to 23% of ICH patients develop hematoma expansion and neurocognitive decline within few hours [90]. MV ICH patients show an inhospital mortality of 90%, while other studies report a 50% of survival at three years, with good functional outcome in only 20% of them [91].

Endotracheal intubation and gas exchange

Intubation followed by MV support is often necessary for providing adequate airway protection and oxygen delivery [92]. Intubation should be considered in the case of GCS $\leq$8 or significant respiratory distress, keeping peripheral oxygenation higher than 92% [93].

Tidal volume and pulmonary pressures

Among respiratory complications, ARDS has been found in 27% of ICH patients, particularly those ventilated with higher V_T, males, patients who received transfusions, higher fluid balance, hypoxemia, acidosis, obesity, smoke, emergent hematoma evacuation, and vasopressor dependence. Higher V_T was identified as the strongest risk factor associated with ARDS in ICH patients [92]. A specific recommendation for ICH patients concerning ventilator management are lacking, albeit current management for stroke patients could be applied also for ICH patients. As suggested by a multicenter RCT on 749 neuro patients (of whom about 40% had a stroke), the application of a lung protective MV strategy, using V_T less than 8 and PEEP of 6–8 cmH_2O, can significantly reduce ventilator-free days and mortality [7].

Weaning and extubation

Extubation failure in ICH patients is around 15%, while external ventricular drainage is more associated with extubation failure than craniotomy or patients' comorbidities [94].

Tracheostomy practice

Predictors for tracheostomy have been evaluated in ICH patients in two retrospective studies. Lower GCS, chronic obstructive pulmonary disease, ICH volume and location, midline shift, intraventricular blood, and hydrocephalus showed a higher predictivity of the need for tracheostomy [95]. Among predictive risk factors for tracheostomy, GCS was detected as the most significant clinical predictor; whereas hydrocephalus (H), septum pellucidum shift (S), and the thalamus location of the ICH (L) were identified as the most useful radiological predictors. Thus, the TRACH score was carried out by assessing the following formula: $3 + (1 \times \text{RS scale}) - (0.5 \times \text{GCS})$, and adding 2 points for L, 1.5 points for H, and 3 points for S [96], to easily identify ICH patients who may require tracheostomy.

Conclusions

In summary, ventilatory management, weaning, and tracheostomy for the main subpopulations of neurocritical care patients is similar to the general population, although specific recommendations and evidences should be taken into account for each specific subgroup, to minimize the risk of respiratory complications and cerebral dysfunction.

Practice points

- Oxygenation and carbon dioxide should be strictly monitored
- A standard tidal volume of 6–8 ml/kg of predicted body weight can be adopted in each brain-injured population
- Zero-PEEP (ZEEP) must be avoided, minimal positive end-expiratory pressure (PEEP) to reach oxygenation targets (SpO_2 >94% – PaO_2 >60 mmHg), recruitment maneuvers can be performed under neuromonitoring surveillance
- A weaning trial should be performed before choosing tracheostomy. The VISAGE score is quite sensitive to predict extubation failure
- Tracheostomy timing is not clear, albeit an early approach might be associated with less risk, to develop VAP and better outcome
- The role of chest physiotherapy in neuro critically ill patients needs to be clarified

Research agenda

- Evidences concerning an optimal approach for ventilatory management in the most common critical care populations are needed
- We need established valid and reliable criteria to be used when selecting patients for tracheostomy and weaning
- Pulmonary rehabilitation can augment the benefits and outcomes of mechanically ventilated critically ill patients, reduce postoperative complications, or improve patient survival following the surgery, but evidences in patients with acute brain injury are urgently needed.

Authors' contribution

DB searched for literature and wrote the manuscript; DSG, GB, IB, NP, PP, and PRMR revised for intellectual contents; CR supported DB in writing the manuscript and revised for intellectual contents. All authors approved the final version.

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Declaration of competing interest

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Abbreviations

AIS	acute ischemic stroke
ARDS	acute respiratory distress syndrome
$BtpO_2$	brain tissue oxygen pressure
CBF	cerebral blood flow
CPAP	continuous positive airway pressure
CPP	cerebral perfusion pressure
FiO_2	fraction of inspired oxygen
GCS	Glasgow coma scale
ICP	intracranial pressure
ICH	intracranial hemorrhage
IH	intracranial hypertension
ISS	injury severity score
ICU	Intensive Care Unit
$PaCO_2$	partial pressure of carbon dioxide
PaO_2	partial pressure of oxygen
PBW	predicted body weight
PEEP	positive end-expiratory pressure
RMs	recruitment maneuvers
SAH	subarachnoid hemorrhage
SAP	stroke-associated pneumonia
SET	stroke-related early tracheostomy
SjO_2	jugular saturation of oxygen
SpO_2	peripheral oxygen saturation
TBI	traumatic brain injury
VAP	ventilator-associated pneumonia
V_T	tidal volume
WFNS	World Federation of Neurological Surgeons

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